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Mitochondria in non-alcoholic fatty liver disease

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ABSTRACT

NAFLD is a common disease in Western society and ranges from steatosis to steatohepatitis and to end-stage liver disease. The molecular mechanisms that cause the progression of steatosis to severe liver damage are not fully understood. One suggested mechanism involves the oxidation of biomolecules by mitochondrial ROS which initiates a vicious cycle of exacerbated mitochondrial dysfunction and increased hepatocellular oxidative damage. This may ultimately pave the way for hepatic inflammation and liver failure. This review updates our current understanding of mitochondria-derived oxidative stress in the progression of NAFLD.

ABBREVIATION SECTION

8-OHdG, 8-hydroxy-2-deoxyguanosine
$\Delta\psi_m$, Mitochondrial membrane potential
AMPK, AMP-activated protein kinase
apoB, Apolipoprotein B